Listing of All Claims Including Current Amendments

- 1. (Currently amended) A method for delivering a peptide into the major histocompatability complex (MHC) class I antigen processing pathway of an antigen presenting cell to elicit a cytotoxic T lymphocyte (CTL) response, comprising contacting said cell with a mutant of E. coli heat labile enterotoxin B (EtxB) or cholera toxin B (CtxB) covalently linked to said peptide, wherein said mutant comprises at least one of the following point mutations within the region spanning amino acid residues E51 to I58 of the β4-α2 loop of EtxB or CtxB: CtxB (E51A), CtxB (Q56A), CtxB (H57A) (SEQ ID NO: 13), and EtxB (H57S) thereby delivering said peptide into said cell.
- (Previously presented) The method of claim 1 wherein the covalently linked peptide is derivable from a protein of interest (POI) or an antiqen.
- (Previously presented) The method of claim 2 wherein the antigen is selected from the group consisting of a viral antigen, a bacterial antigen, a parasitic antigen; and a tumor associated antigen (TAA).
- (Canceled)
- (Canceled)
- 6. (Canceled)
- 7. (Canceled)
- 8. (Canceled)
- 9. (Canceled)

- (Previously presented) The method of claim 1 wherein the mutant comprises a
 point mutation at amino acid residues 51, 56 and/or 57 of the β4-α2 loop.
- (Previously presented) The method of claim 1 or claim 10 wherein the mutant comprises a point mutation at H57A or H57S.
- 12. (Currently amended) A method of preparing a medicament comprising providing a mutant of E. coli heat labile enterotoxin B (EtxB) or cholera toxin B (CtxB) in the preparation of a medicament, wherein the mutant comprises one of the following point mutations within the region spanning amino acid residues E51 to I58 of the β4-α2 loop of EtxB or CtxB: CtxB (E51A), CtxB (Q56A), CtxB (H57A) (SEQ ID NO: 13), and EtxB (H57S) and is capable of delivering an exogenous peptide into the major histocompatibility complex Class I antigen processing and presentation pathways to elicit a cytotoxic T lymphocyte response.
- (Canceled)
- 14. (Canceled)
- 15. (Canceled)
- 16. (Canceled).
- 17. (Currently amended) A method of delivering a peptide to the MHC class I antigen processing pathway of an antigen presenting cell, wherein the method comprises:
 - (i) providing an antigen presenting cell;
 - (ii) contacting the cell with a mutant of E. coli heat labile enterotoxin B (EtxB) or cholera toxin B (CtxB) covalently linked to the peptide; the mutant comprises one of the following point mutations within the region spanning amino acid residues E51 to I58 of the β4-α2 loop of EtxB or CtxB: CtxB

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(E51A), CtxB (Q56A), CtxB (H57A) (SEQ ID NO: 13), and EtxB (H57S) and having GM-1 binding activity; but reduced immunogenic and immunomodulatory activity relative to the corresponding wild type form of EtxB or CtxB: and

- (iii) monitoring for the presence of the peptide in the antigen presenting cell.
- (Previously presented) The method of claim 17, further comprising the step of monitoring the elicitation of a cytotoxic T lymphocyte (CTL) response.
- 19. (Canceled)
- 20. (Canceled)
- 21. (Currently amended) A kit for delivering a peptide to the MHC class I antigen processing pathway of an antigen presenting cell wherein the kit comprises: a mutant of E. coli heat labile enterotoxin B (EtxB) or cholera toxin B (CtxB) covalently linked to the peptide; the mutant comprises one of the following point mutations within the region spanning amino acid residues E51 to I58 of the β4-α2 loop of EtxB or CtxB: CtxB (E51A), CtxB (Q56A), CtxB (H57A) (SEQ ID NO: 13), and EtxB (H57S) and having GM-1 binding activity; but reduced immunogenic and immunomodulatory activity relative to the corresponding wild type form of EtxB or CtxB.
- 22. (Canceled)
- (Canceled)
- 24. (Canceled)
- (Canceled)
- 26. (Previously presented) The kit of claim 21, further comprising means for detecting the location of the peptide in the antigen presenting cell.

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27. (Previously presented) The method of claim 1 wherein said mutant has GM-1 binding activity but reduced immunogenic and immunomodulatory activity relative to the wild type corresponding form of EtxB or CtxB.